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EDITORIAL.

As students of Modern Medicine we find ourselves being taught and trained for general practice by men highly skilled and learned in one or more of the large divisions into which Medicine has been subdivided in its process of evolution. From our humble but necessarily broad viewpoint it sometimes appears that the arbitrary division into Medicine and Surgery in hospital practice is distinctly disadvantageous to the patient. To our way of thinking it would be preferable to send all doubtful cases to a ward or wards staffed by physicians and surgeons who in daily consultation could decide on the best treatment for each individual case. In such a ward one would expect to find many patients with upper abdominal disease or thyroid disease, etc.

This arrangement might lessen the tendency for a public ward patient to be thought of as a case rather than as an individual. Physicians and surgeons would undoubtedly derive mutual benefit by the daily intercourse; and an equilibrium would be reached between surgical haste and medical delay. This is not to be interpreted as a suggestion but is merely the expression of a constantly recurring thought; and as such it is put forward in all humility and sincerity.

The upper years in Medicine fully appreciate the opportunities they have had in recent years of hearing outstanding men from other places. We hope that this practice will continue and indeed that these addresses will become more frequent as time goes on. We wish particularly to express our appreciation to Professor Starr for his interest in this regard. The Journal will be pleased to report such meetings in a similar manner to the report of Professor Wilkie's address in our November issue.

MEDICAL LIBRARIES TO WHICH UNDERGRADUATES IN TORONTO HAVE ACCESS.

1. UNIVERSITY LIBRARY,
2. STUDENTS' MEDICAL LIBRARY,
Medical Building, ground floor, north end.
3. PATHOLOGICAL LIBRARY,
Pathology Building, Second Floor.
4. BIOLOGICAL LIBRARY,
Biology Building.
5. PSYCHOLOGICAL LIBRARY,
Psychology Building, St. George St.
6. SICK CHILDREN'S HOSPITAL LIBRARY,
Floor 2½, H.S.C.
7. LIBRARY OF THE ACADEMY OF MEDICINE,
13 Queen's Park.

A letter of introduction from any member of the Academy admits any student of the upper years to the Library. Dr. Ryerson will supply a letter of introduction on request.

The contents of these libraries will be discussed individually in later issues of the Journal.

GRADUATE STUDIES.

Every student when he enters the course in Medicine wonders when the end is going to be. When the student reaches the final years he knows that study does not end with university life, but in reality only begins, and indeed begins in earnest. What are the relative merits of going right out into practice for oneself, or working under the direction of more experienced men in practice, in hospitals or clinics? There are many branches of special work one may go into also and this has to be decided. What are the best hospitals to go into? What courses have they to offer? How many

months or years should one spend in a hospital? All these questions will appear much more important as the time for graduation draws near. There is also the burning query in the student's mind regarding the manner in which the different hospitals choose their men. Is it by the work they have done during their course in medicine, or is it because one has some "pull" somewhere? Does the hospital which you would like to enter take into account from what University you graduate, and what standing this medical school has on this continent? The relative merits of taking post-graduate work on this continent or going over to the older established schools of Europe, and the money entailed in all of these procedures, are also problems which will interest many of the students. What course did the leading men in our profession follow? Most men have only a relatively short time before they must become money-earners in a community, and the way to place this to best advantage needs expert opinion or suggestion from men who have already gone through the process. An article with an attempt to help the student solve these questions is a difficult thing to compile, but there is an attempt being made to get it into the next issue of this journal.

VOL. IV. OF THIS JOURNAL FREE.

Mr. F. J. Murray, '27, whose article appears in this issue, has been given Vol. IV. The same offer holds for each issue of Vol. IV.; namely, the whole volume free to the writer of the best undergraduate article in each issue, as judged by a member of the Faculty.

TUITION FEES.

In view of the fact that the fees in the Faculty of Medicine in Toronto are to be increased from $150 to $200 next year, the following extract and table from the J.A.M.A., Aug. 28th, 1926, is of special interest.

In the accompanying table the seventy-nine colleges have been grouped according to the amount of fees charged and according to their classification by the Council on Medical Education and Hospitals. Eleven colleges charge fees of $150 or less a year; twenty-seven charge from $150 to $250; thirty-two from $250 to $350; six charge from $350 to $400, and three colleges in New York City charge between $500 and $550.

	Class A	Class B	Class C	P.C.	Total
$ 50 to $100	4	0	0	5.1	4
100 to 150	6	0	1	8.9	7
150 to 200	13	0	1	17.7	14
200 to 250	10	1	2	16.4	13
250 to 300	12	1	1	17.7	14
300 to 350	18	0	0	22.8	18
350 to 450	5	1	0	7.6	6
450 to 550	3	0	0	3.8	3
	71	3	5	100.0	79

Average fee per student in all colleges, $274.

Of the eleven colleges charging $150 or less, ten are listed among Class A (acceptable) colleges by the Council on Medical Education and Hospitals. Among these are the Schools of Medicine of several State Universities for residents of those States.. The five colleges listed by the Council in Class C charge fees of $150 to $350, a year, even though the diplomas from these colleges are reported as not recognized by from 42 to 48 State Licensing Boards.

MISTURA

If you have read anything, even remotely, connected with medicine, which you think others might enjoy reading, Mistura will always be here to let the other readers of this journal know.

The following are recommended by various members of the Faculty and the editors:

Rest and Pain, Hilton.—(Dr. H. B. Van W.).

The Corner of Harley Street, H. H. Bashford.—(Dr. W. F. McP.).

An Epistle, Robert Browning.—(Prof. J. F. McM.), containing the strange medical experience of Karshish, the Arab physician.

A Doctor of the Old School, Ian Maclaren —(W.S.K.). From "Beside the Bonnie Briar Bush."

Rab and His Friends, Dr. John Brown of Edinburgh.—(Prof. A. H.).

Burns.

R. I. HARRIS, M.B.

OUR conception of the problems involved in burns has undergone a profound change during recent years. We now no longer regard a burn as a type of wound, to be dealt with by appropriate local treatment. On the contrary we think of it as a lesion which gives rise to a series of complex pathological processes, the most important of which are general rather than local in their action. Each of these pathological processes corresponds to a clinical stage in the progress of the condition. From this point of view it is possible to divide the clinical course of a burn into five stages. In the order of their occurrence these stages are as follows:

1. Shock.
2. Toxaemia.
3. Sepsis.
4. Repair.
5. Late contractures and deformities.

Naturally not every burn passes through all five stages. A mild burn may be unaccompanied by shock and will never lead to contractures. A severe burn may cause the death of the patient in the stage of shock or in the stage of toxaemia. But in any considerable series of burns the five stages can be recognized, and most severe burns, unless terminating in death, pass through all five stages.

Shock.

The shock accompanying burns is in no way different from surgical shock occuring in other traumatic conditions. The factor which determines its onset and degree is the extent of the burn rather than the depth. It appears within a few minutes after the accident, and is recognized by the usual signs, viz., rapid, feeble pulse, subnormal temperature, low blood pressure, pallor, sweating and thirst. In most cases it can be overcome, and by the end of twelve hours has disappeared. It causes only a small percentage of the mortality due to burns.

Toxaemia.

About twenty-four hours after the accident most severe burns enter into a toxic stage which is peculiar to this type of injury. This is the most highly dangerous period in the course of the disease, since approximately seventy-five percent of the mortality is due to burn toxaemia. The onset is characterized by a clearly defined set of symptoms—high temperature, vomiting, drowsiness, muscular twitchings and convulsions. These apparently are the result of the action of the toxin upon the central nervous system. The action of the toxin is not limited, however, to the central nervous system. Post mortem examination of fatal cases reveals widespread evidence of its effect in the form of focal necroses and petechial haemorrhages in nearly all the parenchymal organs of the body. The post mortem picture resembles closely that produced by diptheria toxin.

The source of the causative toxin has been the object of much research. It has now been established with reasonable certainty that it arises in the burned area, and is distributed to all parts of the body by means of the blood stream. The toxaemia can be reproduced experimentally. Under experimental conditions it can be demonstrated that excision of a burned area, which ordinarily will result in toxaemia will entirely prevent the onset of the toxic stage, while transplantation of the burned skin to an unburned animal will result in the appearance of burn toxaemia.

While the exact mechanism of the production of the toxin is as yet unknown, we may accept the following as a reasonable working hypothesis. The presence of the dead skin excites a reaction in the underlying living tissues which ultimately leads to the complete separation of dead from living cells by a process of granulation. Separation is attended by autolysis of the dead cells along the line of contact by means of ferments derived from the living cells. During this process toxic metabolites are formed which give rise to the period of toxaemia. As the process of separation continues a stage is reached in which the living cells are no longer in direct contact

with dead cells, but are separated from them by a layer of exudate. In consequence digestion of the dead epithelium ceases, and with it the production of toxin terminates. This corresponds to the subsidence of the toxaemia about five days after its commencement. The late appearance of the toxaemia (twenty-four hours after the burn) is due to the manner in which the toxin is produced. Arising as the result of the digestive action of living cells, it requires a period of time for a quantity to accumulate sufficient to produce symptoms.

The chemical nature of the toxin is as yet unknown. There is some evidence that it is protein in origin, and is probably a proteose. In the blood stream the toxin appears to be intimately associated with the red blood cells.

Sepsis.

The stage of toxaemia is followed by a stage of sepsis, whose onset overlaps somewhat the end of the toxaemic period. Infection in some degree is present in every burn in which there has been any considerable destruction of tissue. It is impossible so to treat the burned area by applications as to keep it aseptic. The dead skin, moistened by dressings or underlying secretions, forms an ideal nidus for bacteria, and in it they flourish abundantly. Locally the onset of sepis is evidenced by foul-smelling, purulent discharge, and this is usually accompanied by fever. Septicaemia may occur, and lead to death. The septic state lasts until the sloughs have all separated, and free drainage is established. This usually occurs about the end of the second week. Occasionally the sepis persists even after separation of the sloughs. In such cases amyloid disease is prone to arise. After toxaemia, sepsis is the most important cause of death from burns.

Repair.

With the separation of the sloughs the stage of sepis ends and the stage of repair begins. The burn has now been transformed into a healing ulcer, and its condition is similar to that of any other granulating wound. The stage of repair continues until the wound is completely covered with epithelium. The depth and extent of destruction of epithelium is therefore of extreme importance. From the standpoint of treatment, we may divide burns into two classes: 1. Those of third degree (of Dupuytren's classification), and 2. Those of fourth degree or greater. In the former the epithelium has been destroyed down to the tips of the papillae, so that when the sloughs separate the wound is seen to consist of myriads of tiny ulcers in a sea of epithelium. Healing is rapid, since each tiny ulcer heals from its own margins simultaneously with the rest. In the latter the epithelium over the whole area has been completely destroyed, and epithelialization can only occur from the margins, and hence slowly. Most severe burns are composite and contain areas of both third and fourth degree. When a burn is first sustained it is not possible to determine with any reasonable accuracy the degree to which it belongs. It is not until the dead epithelium has separated that the depth of epithelial destruction can be accurately determined.

Later Contractures.

The stage of late contractures and deformities occurs only in those burns in which the injury has been extensive, of fourth degree or greater, and have healed slowly. Under these circumstances large amounts of to the complete separation of dead from formed later into correspondingly large amounts of scar. The slow contracture of such scar distorts the surrounding structures. If the scar is laid down in the neighbourhood of a joint its contraction produces limitations of movement and deformity of the joint. Similarly when laid down in lax tissues its contraction produces great distortion and disfigurement, particularly in the region of the face and neck.

Treatment.

The clinical stages which have been outlined each constitutes a separate therapeutic problem. Treatment, therefore, can best be discussed under similar headings. In treatment our methods have been profoundly modified as a result of the new conceptions of the pathology concerned in burns, chiefly from the recognition of the state of toxaemia. The most recent advances in treatment have been directed towards the prevention

or cure of the toxaemia, and it is in this field that most advances have been made. The most important of these has been the introduction of the use of tannic acid, which, in addition to the great effect it has upon toxaemia, has so many secondary virtues of value in the treatment of the other clinical stages, that it would appear to be firmly established as the best treatment for burns.

Treatment of Shock.

Treatment of shock in burns is the same as for that of surgical shock. Most cases respond satisfactorily to morphia, heat and fluids. An occasional severe case may require transfusion. Morphia should not be withheld on account of the youth of the patient. There is nothing which can supplant it in the treatment of surgical shock.

Treatment of Toxaemia.

The highly fatal period of toxaemia demands the most careful treatment. The remaining stages present problems which are met with in many other surgical conditions and are dealt with along familiar lines. The toxaemia, in contrast, is peculiar to burns, and is as yet imperfectly understood. It will be necessary to discuss in some detail the steps which have led to our present treatment.

In searching for a specific means of preventing the toxaemia, one naturally thinks first of excision of the burned area. If the toxin is produced in the burned area and only reaches a dangerous concentration after a latent period of twenty-four hours, the rational procedure would be excision of the burned area before any quantity of toxin had been produced. This unquestionably would prevent the toxaemia, as has been demonstrated by experiment. Unfortunately there are certain grave objections to this treatment, which more than counterbalance its benefits. Patients with burns sufficiently severe to cause toxaemia usually have some degree of shock. Early excision would therefore mean the administration of an anaesthetic and an extensive operation upon a shocked patient. Again, at this early stage there is no certain means of distinguishing the exact depth of the burn; in particular of distinguishing between third and fourth degree burns. Were excision undertaken there would be grave danger of converting an extensive third degree burn into an extensive fourth degree wound, and this would enormously enhance the problem of healing. Finally the situation of the burn, as for example upon the face, may be such as to render extensive surgery too mutilating. For these reasons excision of the burned area cannot be entertained.

Exsanguination Transfusion.

Bruce Robertson's observation that the toxin was intimately associated with the red blood cells led him to devise the operation called exsanguination-transfusion. His object was to remove as much as possible of the toxic blood at the height of the toxaemia and replace it with normal blood. The operation, as finally perfected, is carried out in three stages—First a stage in which as much blood is removed from the patient as is compatible with safety; second, a stage in which blood is transfused into the patient while the withdrawal of blood is continued, and finally a stage in which exsanguination is stopped and transfusion is continued until the patient's blood is made up to the volume it was at the beginning of the operation. By this means the patient's circulation is, as it were, washed out. The efficiency of the washing out depends upon the amount of blood exchanged during the first and second stages of the operation, and this in turn depends upon the amount of blood available from donors. In our hands it has been a very important factor in reducing the mortality from burn toxaemia. By its means we have succeeded in reducing death from toxaemia by approximately half. Though it is of great value, it has certain serious limitations which prevent it from becoming universally adopted. It only removes that portion of the toxin which is circulating in the blood, and does not modify in the least the production of toxin at its sources. Consequently the patient may be again inundated with toxin and the operation may require to be repeated once or even twice during the period of toxaemia. The efficiency of the proceeding is dependent upon the interchange of a large quantity of blood during the second stage; three or even five times the estimated total

volume of the patient's blood being not unusual amounts. By using several donors such a quantity of blood cells can be obtained for small children; but for larger children and for adults, the difficulties of obtaining a sufficient quantity of blood are insuperable. Finally the operation is highly technical, difficult and time-consuming, and can only be carried out in a well-equipped operating room and by a team of surgeons thoroughly familiar with the proceedings.

Tannic Acid.

A new plan of treatment which bids fair to revolutionize our results has recently been introduced by Davidson of Detroit. He conceived the idea that it might be possible so to alter the burned skin as to render it insoluble by the enzymes; or at least acted upon with such difficulty that elimination of the toxins would be as rapid as their formation. For this purpose he used tannic acid, the most powerful precipitant of proteins of which we know. The results obtained by the use of this chemical constitute the most marked advance which has been made in years. It reduces toxaemia more successfully than any other measures at our command. In most cases it entirely prevents any serious degree of toxaemia. In a few cases toxaemia does occur even under tannic acid treatment. This we believe to be due to the mechanical difficulty of tanning a deep burn throughout its entire thickness.

Secondary Advantages.

In addition to its marked value in the treatment of toxaemia tannic acid has certain other advantages which of themselves are sufficiently great to warrant continuing its use even though it has no effect upon toxaemia. It has first of all a selective action upon the burned skin. Only the dead skin is tanned. The surrounding living tissue is not affected. Tannic acid precipitates proteins only in faintly acid media: dead tissues are faintly acid, living tissues faintly alkaline, and for these reasons the action of tannic acid is limited to the necrotic tissues. Secondly, for a considerable time it obviates the need for any other dressing. The dead skin is transformed into a black, dry, painless sheet of leather, which effectively protects the underlying living tissues. The tanned area begins to separate about the seventh to tenth day, and until this occurs usually no dressing is required. This freedom from the exquisitely painful ordeal of dressings is something which can be appreciated only by those who have had to deal with numbers of burned patients. Third, many burns are completely epithelialized when the tan separates. This is true of all third degree burns and of some small fourth degree burns. The larger fourth degree burns are, of course, not epithelialized, and present a granulating surface when the tan separates. Fourth, the dry leathery sheet into which the necrotic skin is transformed is a most unfavorable nidus for bacteria. Infection is therefore very much reduced. For these various virtues tannic acid, in all probability, is firmly established as a treatment of burns.

After the trial of various methods of using the tannic acid the following has been found to be simple and effective. As soon as possible after admission of the patient the burned area is cleaned with gasoline or ether in order to remove the greasy or oily application which usually has been applied. The vesicles are opened and the loose skin removed. The burned area is then sprayed with a freshly prepared 10 per cent. aqueous solution of tannic acid from an ordinary atomizer. It is important that the solution be freshly prepared, since in standing the tannic acid changes to the much less active tannous acid. The spraying is repeated every half hour for the first 24 hours, by the end of which time the dead skin is thoroughly tanned. At each spraying a thin film only is spread over the burn since any greater quantity will only run off. During the period of spraying and afterwards the patient lies in bed unclothed and without bed clothes. The surrounding air is heated by electric lights under a canopy.

Treatment of Sepis.

In the treatment of the stage of sepis, the most important factor is the removal of the necrotic skin. While this is adherent to the underlying tissues it remains a breeding ground for bacteria which no form of antiseptic dressing can effectively combat. The use of tannic acid by converting the

necrotic skin into dry parchment like leather has greatly reduced the sepis. Even here, however, if the burn is of fourth degree, moisture collects beneath the tan and becomes infected. It is of great importance therefore to remove the tan in order to establish free drainage. For this reason the burn should be examined daily after the first week, and all loose slough removed with scissors or scalpel. As soon as the sloughs begin to separate antiseptic dressings are probably of some value reducing the infection though they can never completely prevent it. After the tan is entirely separated the granulating wound can be cleansed by the use of moist antiseptic compresses. We have found the chlorine antiseptics (eusol and Dakin's solution) by far the most valuable.

Treatment of Stage of Repair.

Treatment during the stage of repair resolves itself into the treatment of a healing ulcer. The infection should be reduced to a minimum by means of antiseptic dressings. As soon as the wound is clean a decision must be reached as to whether skin grafting will be necessary. All extensive fourth degree burns should be covered with Thiersch grafting as early as possible. Scar production is thus greatly diminished. The grafts should be taken from the patient. We have had no success with grafts from donors, nor with whole thickness grafts. Pressure in some form is essential for successful grafting. For this purpose paraffined mesh gauze sutured to the margins of the wound is valuable. An effort should be made during this stage to minimize the extent of the late deformities by maintaining the joint in positions of greatest usefulness.

Treatment of Contractures.

In a certain number of cases, in spite of the greatest care, the amount of scar tissue which is laid down will produce late contractures and deformities. The only satisfactory method of treatment for these is removal of the scar. Sometimes this can be obtained by excision of the scar and suture of the margins after undermining. More frequently recovering of the area from which the scar has been taken involves transplantation of skin and subcutaneous tissues from other parts of the body. This must be done by means of pedicled skin flaps.

Volkmann's Ischaemic Contracture.

H. R. ELLIOTT, 27.

THIS condition was first described by Volkmann in 1857. It may be stated to be a contracture of the fingers and sometimes of the wrist, which comes on rapidly, with loss of power, which is not absolute, in the forearm muscles, following upon too tightly bandaging the arm. It is most commonly seen in cases of fracture, in the neighbourhood of the elbow joint and within the age limit of from one to fourteen years.

Etiology.

When Volkmann described this type of paralysis following the application of a constricting bandage, he expressed the opinion that it was due to the obstruction of the arteries, and anaemia of the muscles of the constricted extremity.

Leser was of the same opinion and believed that the accompanying venous stasis merely hastened the development of the pathological condition. He states that the deprivation of the muscles of oxygen resulted in the degeneration of the muscle fibres, and later their absorption. Both he and Volkmann recognized that the paralysis and contracture were associated with marked manifestations of inflammation.

Bardenheur believed that the pathological changes were due to a vascular disturbance in the nature of venous stasis, and, that the degeneration of the muscle fibres was due to toxic metabolic products which are retained in the muscle.

J. P. Murphy believed the condition due to an effusion of blood and serum into injured muscles. He noted that the Radial

pulse was present throughout the entire course in some cases.

This observation of Murphy's threw some doubt on the Volkmann Theory. Further doubt is thrown on Volkmann's idea of arterial blockage, by the fact, that in Halstead's collection of instances of ligation of the Common Iliac artery, no cases of actual paralysis are found. Furthermore, paralysis of muscles does not accompany progressive obliteration of arteries, even when occlusion is so extreme as to cause gangrene.

It remained for Brooks of St. Louis, through a great many experiments carried on in 1921-22, to definitely decide the etiological factor as being that of venous stasis. In his first group of experiments, tourniquets, splints, and plaster bandages were used. These were applied to the foreleg of a dog, above the elbow, but, because of the difficulty in separating the effect of direct pressure on the nerves, and the circulatory disturbances of the tissues, as causes of the paralysis, this method was abandoned. He next tried ligating the main artery of supply, and in these cases where the animal did not die or develop gangrene, the recovery was complete and the muscles did not show pathological change. In many of these cases he was able to demonstrate the presence of a collateral circulation, and in order to overcome this factor, he finally devised a plan of experimenting on an isolated muscle. For these experiments the Rectus Femoris muscle was chosen, on account of its accessibility, and the fact that its circulation is supplied by a single artery and vein, which are easily isolated. Its nerve supply is a single branch from the Femoral Nerve. The operative technique was the same for all the experiments. The dog was anaesthetized and the Rectus Femoris exposed by an incision made on the anterior surface of the thigh and distal third of the abdomen. The muscle was then freed from the surrounding tissues everywhere, except at its origin and insertion. The artery, vein, and nerve were completely isolated from one another. A tight ligature was placed about each end of the muscle, thus giving a muscle preparation completely isolated from all vascular connections, except a single artery and a single vein. The nerve supply was intact and the circulation in the surrounding tissues was normal. When the nerve alone was severed complete paralysis of the muscle followed, but there was no evidence of inflammatory reaction or fibrosis. He then tried injecting a quantity of blood into the isolated muscle, leaving the blood and nerve supply intact. Later, on examining the muscle he found nothing abnormal in physiological reactions, nor was there any evidence of inflammation or fibrosis. When the artery was ligated, leaving the vein and nerve intact, the muscle retained wholly its physiological and anatomical characteristics, or became completely necrotic and disappeared. In no instance did he find that the anaemia resulted in an inflammatory reaction. In those cases in which the muscle remained viable, Brooks expresses the view that the muscle must have received nutrition from the surrounding tissues, or by a reverse circulation in its vein, until the time of establishment of a new circulation, by vessels growing in from the surrounding tissues. Of four experiments in which the artery and vein were ligated leaving the nerve supply intact, two showed no pathological changes, whereas in the other two, the muscle became necrotic and disappeared. The fact that muscles often remain normal when both artery and vein are ligated, was demonstrated also during the War, where in cases requiring ligation of a main artery, it was found safer to ligate its accompanying vein along with it, than to ligate the artery alone. When, however, the vein alone was ligated, leaving the artery and nerve intact in every instance marked pathological changes were found. Within an hour after operation the muscle became swollen, hard, and dark blue. Stimulation of a muscle or nerve at this time usually gave a slight contraction. At the end of twenty-four hours there was a tremendous extravasation of blood into the muscle. At forty-eight hours there was a marked infiltration of polymorphs, and at this time many muscle fibres were clearly necrotic. In four days there was a marked proliferation of connective tissue, and extensive degeneration of muscle fibres. This connective tissue was found in between the muscle fibres and

replacing the necrotic ones. Contractures of the muscles were always present.

Pathology.

The above description given by Brooks fits in very nicely with an account of another observer, of the pathological changes observed in a piece of muscle removed from a patient, suffering from Volkmann's Ischaemic Contracture of six weeks' duration. In the gross the muscle is much paler than normal and feels very firm. Microscopically there is evidence of an acute inflammatory reaction manifested by a marked leucocytic infiltration. There is also a marked growth of very cellular connective tissue which, in many cases completely replaces the muscle fibres. Other observers describe some evidence of degeneration in that there is loss of muscle nuclei and the muscle fibres themselves appear somewhat smaller. There is no evidence, however, of fatty degeneration.

Symptoms and Signs.

The chief symptom and one that should always be kept in mind is that of pain. This comes on a few hours after the splints are applied, and at first is of a curious tingling character. Soon, however, it becomes very severe and is most unbearable to the patient. For this reason, morphia should not be used when "putting up" a fracture about the elbow joint, in fact, a patient with a properly set fracture in any location does not require morphia. Two chief signs which accompany the pain are swelling and cyanosis of the fingers, accompanied by inability to put the fingers through a full range of movements. This last sign is most important, and to rule out the possibility of a developing contracture it must be A FULL RANGE OF MOVEMENT, because in some cases at this stage, the patients are able to flex and extend the fingers to a limited degree at the metacarpal-phalangeal articulations. Along with the above there may be some disturbance of sensation; that of light touch may be impaired or absent from the whole palm, or from only that area supplied by the median nerve. If the splints are removed at this time the flexor muscles of the forearm are found to have a curious, hard brawny feeling. If these signs are not recognized and the condition attended to immediately, contracture and weakness follow rapidly. In the early stages the contracture shows itself in the form of marked flexion of the second and third phalanges, with extension at the metacarpal-phalangeal joint. As the contracture progresses the deformity is exaggerated and there is added an increasing flexion of the wrist and a slight flexion of the elbow in semi-pronation. The thumb is often firmly adducted.

Diagnosis.

The simultaneous appearance of paralysis and contracture are of great diagnostic value, because this distinguishes the condition from contractures following peripheral nerve lesions or those of Central Nervous System, in which paralysis precedes contracture by a long interval. The muscles in most cases retain normal electrical reactions. In this condition attempts to extend the wrist passively cause marked flexion of the fingers; full flexion of the wrist permits extension of the fingers.

In late Ulnar paralysis with "main en griffe," although it may simulate this condition the muscles affected are those supplied by this nerve alone. There may also be sensory and trophic disturbances of the skin. Also in Ulnar paralysis the patient is unable to separate and approximate the fingers of the affected hand, whereas in Volkmann's contracture a condition which does not affect the intrinsic muscles of the hand he is able to do this, thus demonstrating an intact ulnar supply to the interossi muscles.

Median nerve paralysis can hardly be confounded with this condition.

In Radial nerve paralysis, although we have drooping of the wrist, it is due to paralysis of the extensors and not contractures of the flexors. The flexion here can easily be overcome by passively extending the wrist.

Prognosis.

This should be guarded and is usually bad. Inasmuch as it is impossible to determine clinically the exact amount of damage, an accurate prognosis cannot be given until the reaction to proper treatment has been watched for a time.

Treatment.

PROPHYLACTIC.—In children one to fourteen years of age with fractures about the elbow region, tight dressings should be avoided as well as an extreme angle of flexion at the elbow. The radial pulse should be felt coming through at the wrist. In all injuries of this type the medical attendant should be in constant touch, so that if undue pain, swelling and cyanosis occur, or if the patient is unable to move the fingers through a normal range of movement prompt measures may be taken to prevent the full establishment of the lesion.

IMMEDIATE.—All dressings should be removed, the limb elevated and gentle massage and passive movement given, while the site of fracture is steadied and supported by the hand. If in spite of these measures the swelling and cyanosis increase, one should attempt decompression of the flexor muscles, by splitting the deep aponeurosis longitudinally either subcutaneously or together with the skin if that is oedematous and shiny. Sterile dressings and a light supporting splint with the elbow at a right angle should be applied. When the danger of the contracture is passed the fracture may be treated as best circumstances permit.

LATE.—As has been stated elsewhere, the essential lesion consists in the change of the muscle to fibrous tissue. If this change involves all the muscle fibres, no treatment, operative or otherwise, can ever restore active motion to the damaged muscle. If, however, the damage involves only a portion of the muscle, the remainder is potentially able to cause voluntary movement, provided it can be freed from the handicap of the contracted cicatricial tissue mingled with it. Thus the best treatment will be the best means of removing this handicap.

Stretching or tearing the contracted muscles under an anaesthetic has been proven useless. Multiple tenotomies cures the deformity but involves a permanent disability. Two types of operation are in use to-day: the one a tendon lengthening, where all the flexor tendons are lengthened in order to permit the simultaneous extension of the fingers, hand and wrist to a straight line, the other an osteotomy which shortens the radius and ulna sufficiently to allow the same result. In connection with these operations it must be remembered that the limb is in a poorly nourished condition, that in the osteotomies the growth of the bones may gradually cause a recurrence of the deformity and that the lengthening of a muscle tendon lessens the muscle's efficiency.

Most cases to-day are treated by plaster of Paris splints. This combined with physical therapeutics obtains results which for promptness and quality are as good or better than those obtained by operation. This method consists in the persistent use of splints in the form of "wedging plasters," applied so as gradually to stretch the contracted muscles. First the limb is put up with the wrist fully flexed and the fingers straight, though bent at the metacarpal-phalangeal articulations. By slow degrees the fingers are extended at this latter joint by means of wedges placed in the plaster. The next consists in gradually extending the wrists in the same manner with the hands straight, and when once the deformity is overcome a system of exercises designed so as to prevent the recurrence of the deformity are instituted.

The Aetiology of Anaemia and Its Importance in Diagnosis and Treatment.*

DUNCAN GRAHAM, M.B.

THE careful correlation of symptoms and signs will ever remain the physician's most dependable guide in the diagnosis of disease. It is only with this information that more modern methods of diagnosis can be intelligently applied for the further elucidation of the nature of the condition present. Among the common signs of disease presented by patients is pallor. In certain cases its association with other signs or with certain symptoms, as in Addison's anaemia, makes a correct diagnosis reasonably certain. More often it is associated with symptoms present in a number of conditions, as weakness and lassitude, and an accurate diagnosis is impossible without further investigations. In this type of case the physician, having decided from an examination of the skin and mucous membrane that the pallor is due to a deficiency of haemoglobin, too often prescribes a tonic of iron and arsenic without a further investigation into the exact cause of the anaemia. As a result, many opportunities are lost for the application of more effective treatment.

Probably the chief factor to this all too common practice of the symptomatic treatment of anaemia is our lack of knowledge of the process at work in its development. Even with our present inadequate knowledge of the subject it appears quite clear that anaemia results from three main causes: loss of blood from haemorrhage, as in duodenal ulcer; increased destruction of red blood cells, as in haemolytic jaundice; or deficient formation, as in benzol poisoning. In every case of anaemia an attempt should be made to ascertain which or what combination of these main factors are responsible in its production and then determine, if possible, the more exact aetiology of the condition. Among important aetiological causes may be mentioned: haemorrhage, external or internal; infection, malignant disease; intestinal parasites; chemical poisons; and unknown haemolytic agents. By a careful history and physical examination it is usually possible to determine whether or not haemorrhage is the cause of the anaemia. From the appearance of the skin and conjunctiva, by the examination of the urine for urobilin, of the blood serum for the presence or absence of bilirubinaemia, as shown by the van den Bergh test, and from the haemotological characteristics of the blood, information is obtained which indicates the presence or absence of an anaemia due to increased destruction or to deficient formation of blood. In the examination of a blood smear the presence of red blood cells showing polychromosia and reticulation indicates immature cells and is a sign of regeneration on the part of the bone marrow. The absence of these cells in anaemia is good evidence of deficient blood formation.

Anaemia, the result of loss of blood and that following toxic doses of benzol, has been studied most carefully, both experimentally in animals and clinically in man. It may be of value for our present purpose to review some of these findings, as the information gained from these observations is of the greatest value in the better understanding of anaemia due to other agents. The site of formation of the red blood cells in adult life is the bone marrow, mainly the marrow of the ribs and the ends of the long bones. From this source the circulating blood is maintained normally with a fairly constant number of mature red blood cells. Here these cells live and function for about thirty days. Old and dying cells are being constantly removed, chiefly by the spleen, and replaced by mature ones from the bone marrow. If a small quantity of blood is lost by haemorrhage, internal or external, it is rapidly replaced by normal cells; with a more severe haemorrhage of 400c.c. or over, the loss is replaced in part by immature cells, some of which may be nucleated. After a

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short time, usually within three weeks, the immature cells disappear from the circulation, and normal conditions are re-established. Should the haemorrhage be repeated ever so often after immature cells disappear from the circulating blood, the anaemia produced is soon recovered from. But if it is repeated sufficiently often without allowing the blood to return to normal, the bone marrow becomes fatigued, as evidenced by the absence of immature cells in the circulating blood, and anaemia results from deficient blood formation, in addition to loss from haemorrhage.

In benzol poisoning an anaemia develops which is characterized by depressed blood formation from the onset. Examination of the blood shows a diminution in the number of red blood cells, the percentage of haemoglobin, absence of immature cells, decrease in platelets and in the number of granular leucocytes of varying degree dependent upon the severity and duration of the poisoning. As these effects are present from the beginning and affect the formation of all types of blood cells in the bone marrow, benzol may be considered as an agent primarily affecting the bone marrow, causing depressed formation, and, finally partial or complete aplasia. The skin and conjunctiva are white; the urine is pale—urobilin absent; the van den Bergh test is negative—absent bilirubinaemia; the case shows all the characteristics of an anaemia due to depressed blood formation. Owing to the extensive use of benzol in industry, this type of anaemia is seen more often than formerly.

A similar type, commonly referred to as idiopathic aplastic anaemia,, occurs. Although the cause is at present unknown, the knowledge gained from the study of cases of anaemia following benzol poisoning makes it quite clear that the condition arises from the action of an unknown poison on the bone marrow, causing deficient formation of red cells, and later aplasia.

Although it is possible to produce experimentally by repeated bleedings at appropriate intervals an anaemia due to deficient blood formation, this rarely, if ever, occurs clinically; and where such a type of anaemia exists in a patient giving a history of haemorrhage, another cause must be looked for. It is not uncommon in cases of duodenal ulcer giving a history of repeated bleeding to find an anaemia of this type. Further investigation usually establishes the presence of a focal infection in teeth or tonsils as the probable cause of the depressed condition of the bone marrow. In pulmonary tuberculosis the toxic products of the tubercle bacillus affect the bone marrow, depressing blood formation, and an anaemia of varying degree results. Should a haemorrhage occur, the severity of the anaemia is increased, but the marrow does not respond in a normal fashion with signs of active regeneration. The condition of the marrow at the time of the haemorrhage determines its reaction to the loss of blood.

In certain anaemias the skin and conjunctiva have a yellowish tint; the urine contains varying amounts of urobilin; the blood serum gives a positive, indirect van den Bergh test—bilirubinaemia is present; and the examination of the blood smear shows the presence of immature red cells. This combination of findings has come to be recognized as due to increased blood destruction. The best example of this type of anaemia is congenital or acquired haemolytic jaundice, a condition further characterized by increased fragility of the red blood cells. Following splenectomy in this condition there is a disappearance of the urobilinuria, the bilirubinaemia, the yellowish colour of the skin and conjunctiva, and of immature red cells from the circulating blood. From this result, one may conclude that increased blood destruction is a factor in the production of anaemia, and that in haemolytic jaundice the cause of the increased destruction is located in or related to the spleen, and can be controlled by removal of this organ.

A common type of anaemia characterized by increased blood destruction is Addison's anaemia—commonly called pernicious anaemia. During a relapse all the findings indicating increased blood destruction, as found in haemolytic jaundice, together with haemoglobindaemia are present. These tend to disappear during a remission and to reappear in the next relapse. Although all cases show signs of increased destruction of varying degree in the beginning, deficient

production, or aplasia of the bone marrow, may be the process active at the end in a certain number of cases. In these, relapses are usually not so sharply defined, and tend to occur at longer intervals. The reaction of the bone marrow to increased blood destruction in Addison's anaemia is quite different to that in haemolytic jaundice. The chief difference is the presence in the circulating blood of numbers of red cells larger than normal and possessing a normal haemoglobin content. This is the earliest and most characteristic haematological finding in Addison's anaemia. It probably indicates an abnormal blood formation on the part of the bone marrow. Similar haematological changes are found in certain cases of anaemia associated with the presence of dibothrocephalus latus in the intestine, and have been reported as occurring very occasionally in anaemia associated with pregnancy, syphilis and malaria. As recovery from the anaemia follows the expulsion of the worm from the intestine, the emptying of the uterus in pregnancy, or the successful treatment of the existing syphilitic or malarial infection, it may be questioned whether one should consider these possible causes of the Addison type of anaemia. At any rate, the presence of dibothriocephalus latus in the intestine, pregnancy, syphilis and malaria must be considered as causes of an anaemia due to increased blood destruction, having signs and symptoms closely simulating Addison's anaemia. Occasionally in malignant disease, especially carcinoma of the stomach, an anaemia indistinguishable from Addison's may be present. In these rare cases it is more than likely that the patient is suffering from two conditions. In the vast majority of cases of malignant disease the associated anaemia can be easily differentiated from Addison's anaemia by a careful blood examination.

Addison's anaemia is characterized by certain signs and symptoms which indicate that it is not a disease of the bone marrow alone. In fact, it might equally well be considered as a disease of the nervous or digestive systems. The aetiological agent, whatever it may prove to be, affects the nervous and digestive systems, producing characteristic changes in addition to those that are the direct result of the anaemia itself. Signs and symptoms indicating disease of the nervous or digestive systems are present in every case. They may dominate the clinical picture and be present for months, or even years, before signs and symptoms of anaemia develop. In many instances patients come complaining of numbness or tingling of the fingers, difficulty in doing fine work or picking up small objects, difficulty in walking, signs and symptoms due to disease of the posterior or lateral column of the cord—combined degeneration. Common gastrointestinal symptoms are periodic sore tongue, diarrhoea, constipation, or alternating constipation and diarhoea, anorexia, nausea and vomiting. Seldom are all these signs and symptoms present in one case, but some combination of them always occurs. Evidence that the bone marrow is involved in these cases, whether signs of anaemia be present or not, is strongly suggested by the fact that the blood almost always, if not invariably, shows the characteristic macrocytosis diagnostic of Addison's anaemia.

In severe infections caused by streptococcus haemolyticus and staphylococcus aureus, a severe grade of anaemia may rapidly develop from increased blood destruction. A similar, but less severe, anaemia is present in acute rheumatic fever. The anaemia in chronic infections may be due to increased destruction in the beginning, but the anaemic condition is maintained from deficient blood formation. This very common type of anaemia is usually caused by malignant disease, exposure to chemical poisons, mainly phenol, benzol, and anilin derivatives, and intoxications as in nephritis, in addition to chronic infections. Improper diet and bad hygienic surroundings, conditions which affect the general health of the individual, tend to depress blood formation, resulting in a mild grade of anaemia.

Apart from congenital or acquired haemolytic jaundice, idiopathic purpura haemorrhagica and Banti's disease, where splenectomy produces such gratifying results for the cure of anaemia, no specific methods of treatment are available beyond an attempt to remove the aetiological cause. Accuracy in diagnosis is essential to any success in treatment. As anaemia is not the result of prim-

ary disease of the bone marrow in the majority of cases, but develops as a secondary manifestation in diseases primarily affecing other organs or systems of the body, the course of the anaemia is usually dependent upon our success in the treatment of the primary condition. In the group of cases where the chief manifestations of disease are the signs and symptoms of anaemia, an attempt must be made by thorough investigation to determine: firstly, whether it is the result of haemorrhage, increased destruction or deficient formation of blood; secondly, the character of the reaction of the bone marrow by a haematological examination; and, thirdly, a consideration of possible aetiological agents that may be responsible in the production, such as: haemorrhage, internal or external; infection; malignant disease, chemical poison; and intestinal parasites. With this information and care in investigation, fewer cases of apparently slight anaemia, but resulting from a severe condition, as carcinoma of the stomach, subacute bacterial endocarditis, or duodenal ulcer, will be left undiagnosed as anaemias of unknown origin, and our respect as physicians for the sign pallor as a warning of disease demanding our best attention will be greatly increased.

Respiration Catalysts.

MISS J. McFARLANE, M.A., DEPT. OF BIOCHEMISTRY.

THE living organism continually generates heat and energy for the normal functional activity of the body, and this energy is derived for the greater part from the oxidation reactions in the cells. These are accompanied by an absorption of oxygen into the tissues and a giving off of carbon dioxide.

Lavoisier defined respiration as "a slow combustion of carbon and hydrogen which is similar in all respects to that which takes place in a lamp or lighted candle." Recent work has shown oxidations to be reactions much more complex than they were once thought to be; that even the union of hydrogen and oxygen to form water is not a direct union, but involves a series of reactions. Similarly in the combustion of carbon monoxide, intermediate products such as formic acid and hydrogen peroxide have been isolated. On the assumption that no fundamental differences exist between oxidations in living tissues and those in vitro, the problem of the mechanism of respiration becomes formidable when we consider the real complexity of the apparently simple reactions referred to.

The Nature of Oxidation.

Oxidation is defined as the process in which a substance acquires oxygen, and the oxidation of one substance always implies the reduction of another. A substance is also said to be oxidised if hydrogen is withdrawn from it. This process of dehydrogenation is illustrated in the reaction of hydrogen sulphide with ferric chloride,

$$2FeCl_3 + H_2S = 2FeCl_2 + 2HCl + S.$$

In terms of the electrolytic dissociation theory, oxidation becomes even more comprehensive, not only including processes involving transfers of oxygen or hydrogen, but also other processes in which neither oxygen or hydrogen are transferred, but in which there are transfers of ionic charges. If an ion in a reaction gains positive charges or loses negative charges it is said to be oxidised. It is reduced if it gains negative charges or loses positive charges. The reaction between ferric chloride and potassium iodide solutions serves to illustrate this type. Ferric chloride is reduced by potassium iodide and ferrous chloride, potassium chloride and iodine are formed. The reaction in terms of the ionic theory is expressed in the following equation:

$$\overset{+++}{Fe} + \overset{---}{3Cl} + \overset{+}{K} + \overset{-}{I} = \overset{++}{Fe} + \overset{---}{3Cl} + \overset{+}{K} + I.$$

The ferric ion is the oxidising agent, losing one positive charge which is taken by the iodide ion, neutralizing the negative charge it carries. The iodide ion is thus the reducing agent. Similarly, many oxidation reactions, could we trace them out, would be found to involve the transfer of positive or

negative charges from one element to another. Oxygen itself acts as an oxidising agent because of its great tendency to remove negative charges from other substances.

Theories of Oxidation.

All chemical processes in the cell are regulated for the production of quick response to a stimulus and quick recovery from the effects of a stimulus. Glucose or amino acids in sterile solutions remain in contact with oxygen without undergoing change, yet if absorbed into the tissues of a plant or animal they undergo rapid decomposition accompanied by an absorption of oxygen and an evolution of carbon dioxide. The problem of biochemical oxidations is the discovery of the factors promoting the oxidations of these materials at such low temperatures and with such high velocities. Is the explanation to be found in the high reactivity of the oxygen in tissues? It is known that the chemical reactivity of hydrogen and oxygen varies, that it is possible to obtain hydrogen having a wide range of reducing power, according to the metals used in generating it. Oxygen exists in different states of oxidation; the hitherto recognized forms such as ozone and atomic oxygen represent only the more stable forms. If oxygen is utilised in a very reactive form, what mechanism in cells renders the element in this active state? As a result of searches in the tissues of plants and animals for substances of high oxidising activity, considerable evidence has accumulated which has established the two theories generally accepted.

The theory of direct oxidation by active oxygen is illustrated by the following experiment. A solution of gum guaiac, containing guaiaconic acid does not become oxidised on exposure to air, and the addition of hydrogen peroxide produces no change. If now a little blood, or an extract of horse-radish root be added, the guaiaconic acid becomes blue due to oxidation. The blood and the plant juice contain a catalyst, a peroxidase, which activates the oxygen of the peroxide, liberating it in a form which readily combines with guaiaconic acid. In a similar manner, amino acids, lactates or glucose mixed with peroxide, are partially oxidised if a ferrous salt is added to catalyse the reaction.

If guaiacum solution is dropped on the cut surface of a potato out of contact with air, no change in colour occurs. On being exposed to air the spot becomes blue without the addition of hydrogen peroxide. It is thought that the potato contains an autoxidisable substance which becomes a peroxide on exposure to air. The potato contains too a peroxidase which releases active oxygen from the peroxide compound, this oxygen being absorbed by the guaiaconic acid. This mechanism in the potato is said to be an oxidase system, including the autoxidisable peroxide-forming substance, and the peroxidase catalyst. Such oxidase systems are largely responsible for plant oxidations, and for the oxidation of certain metabolites in animal tissues.

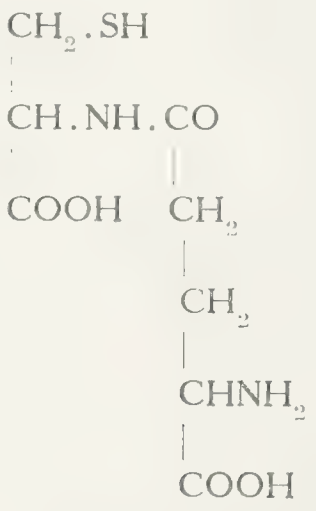
Dehydrogenation theories, on the other hand, involving the principle of the activation and removal of hydrogen from the substance to be oxidised may be illustrated by the experiment in which carbon monoxide is oxidised to carbon dioxide. Water is essential for this reaction which has been proven to occur in the stages represented by the following equations:

$$CO + H_2O = HCOOH$$
$$HCOOH = CO_2 + H_2$$
$$H_2 + O_2 = H_2O_2$$
$$H_2O_2 = H_2O + 1/2\ O_2$$

Formic acid, hydrogen and hydrogen peroxide have been isolated as products, and the extraordinary observation is, that although water is an essential element in the reaction, oxygen is not. The carbon dioxide is formed as a result of the dehydrogenation of the formic acid, and this is the essential change. This reaction occurs in the **absence of oxygen** if water is present, and if the hydrogen is absorbed by a suitable hydrogen acceptor such as palladium black, which functions also as a catalyser. Similarly glucose may be oxidised to carbon dioxide in the absence of oxygen, and succinates in animal tissues are dehydrogenated catalytically by means of enzymes occurring in the cells.

The Mechanism in Animal Tissues.

The respiratory function in animal cells is thus based on these oxidation and dehydrogenation reactions which are catalysed by certain soluble enzymes termed peroxidases, or by haemoglobin of the blood, or by certain insoluble enzymes in tissues which are chiefly dehydrogenases. Various substances which are autoxidisable have been isolated from tissues. Among these are unsaturated fatty acids, and certain lipoids containing these, and other organic compounds which contain the sulphydryl group in their structure. Cysteine and glutathione are examples of this latter class. It was the evidence of a catalytic dehydrogenation mechanism in tissues which led Professor Hopkins of Cambridge, to make the discovery of glutathione. This substance is a dipeptide of cysteine and glutamic acid and it can be readily synthesized from these amino acids. It occurs, widely distributed, in animal tissues and in yeast, and it has been isolated from yeast and from the muscle, liver, and red blood cells of animals. In these tissues it occurs in the form which has the following structural formula:



The autoxidisation of this substance in an alkaline solution produces a disulphide, and if the reduced form is represented as G.SH, the oxidation reaction could be shown thus:

$$2G.SH = G.S\text{-}S.G + 2H$$
$$2H + O_2 = H_2O_2$$

Glutathione in the presence of a peroxidase could form an oxidase system. Moreover, in the tissues this substance is autoxidisable, and the oxidised form, the disulphide, can oxidise tissue substances catalytically by removing hydrogen from them. By so doing the glutathione becomes reduced and assumes again the role of an autoxidisable cell constituent. Professor Hopkins has succeeded in oxidising unsaturated fatty acids of lecithin and linseed oil, and certain protein metabolites in tissues. However, the relation of glutathione to the respiratory function has not been completely demonstrated as yet, although it has a significant role in cell processes, its activity accounting for about one-tenth of the total oxygen absorption of a tissue. In the cell it is in equilibrium with oxygen and with certain oxidisable metabolites and the efficiency of this mechanism depends on the property of glutathione as an autoxidisable cell constituent, and also as a normal hydrogen acceptor.

The Significance of Iron in the Oxidation Reactions.

Dr. Otto Warburg (Berlin) claims that the rapid autoxidation of substances like cysteine and glutathione is not a property of the substance itself. He has proven by careful experiments that 99% of the oxidation rate of cysteine is due to impurities in it, chief among these being salts of iron. A sample of cysteine prepared by Dr. Warburg from purified chemicals in contact with only pure quartz utensils, will absorb only from 1/100 to 1/150 of the amount of oxygen which will be taken up by cysteine prepared according to the usual methods and in ordinary glassware. The addition of iron chloride to this pure cysteine, in amounts so minute that they cannot be detected by any of the usual tests for iron, will increase the oxygen absorption to a marked degree. Harrison's recent work on glutathione confirms Warburg's theory, and proves that the catalysis in the glutathione system is a property of a dipeptide-iron complex.

The relation of iron salts to oxidation processes has long been known. Haemoglobin has a peroxidase reaction and is easily replaced in the reaction by simple iron salts. These iron salts will replace blood in certain bacterial cultures which will not thrive anaerobically without it. Crystals of magnetic ferric iron have been used successfully in such cultures. Iron salts accelerate the action of tissue oxidases and this

metal is found in the ash of many oxidising enzymes. Iron salts are not all active peroxidases; ferricyanides and ferrocyanides are inactive, and both ferrous and ferric iron have active and inactive salts. Only a trace of the salt is needed to give the reaction, and complex iron salts such as sodium pentacyanoaquoferriate, $Na_2\left[\begin{matrix}\cdots\\ Fe\ OH\\ (CN)_5\end{matrix}\right]$ and sodium pentacyanoaquoferroate $Na_3\left[\begin{matrix}\cdot\cdot\\ Fe\ OH_2\\ (CN)_5\end{matrix}\right]$ will give the peroxidase reaction in dilutions of 1:500,000. Dr. Oscar Baudisch has observed the peroxidase reaction of ferrous carbonate in certain mineral waters just as they emerge from the ground. The water soon loses the property on standing, especially if exposed to sunlight or if heated. Magnetic ferric oxide crystals are catalytically active, but their peroxidase reactivity and also the magnetic properties of the crystals are lost after heating to 550·C, although no chemical change in the crystals has occurred. Baudisch suggests a relation between the catalytic property of iron and the particular arrangements of its atoms in the crystal or in space.

Recall the fact that life without iron is impossible, and that iron occurs in all living cells in minute amount, perhaps not more than 1/000 to 1/000 milligrams per gram of cell substance. Manganese and other metals are found in addition to iron, but their reactivity is less and their quantity is infinitely small. Warburg and Meyerhof in studying the respiration of sea urchins' eggs, were able to increase the respiration, measured by the absorption of oxygen, by increasing the amount of iron salt in the egg material. Similarly the addition of traces of iron to cysteine and glutathione or to lecithin or muscle tissues, greatly accelerates the absorption of oxygen by these substances.. If now a cyanide is added to the sea urchins' egg material, in amount just sufficient to combine with the iron contained in them, the oxygen absorption is inhibited. The addition of small amounts of cyanide to sulphydryl compounds also prevents their oxidation. Further it has been proven that these sulphydryl compounds have a very slow rate of oxidation if they are prepared free from iron and it is therefore reasonable to assume that iron in some specific chemical or physical state is a catalyst in oxidations promoted by cysteine or glutothione, and is the mechanism in operation in the sea urchins' eggs.

Warburg's Model of Cell Respiration.

Dixon and Tunicliffe have investigated the effect of various factors on the rate of oxidation of glutathione, and have found a greater absorption of oxygen if the glutathione solution is in contact with the surface of pulverized glass or platinum. Oxidation reactions in cells probably occur on the surface of structural elements in the cytoplasm because a complete disintegration of structure tends to destroy the respiratory power. For example, if birds' blood is hemolysed and centrifuged the nuclei and granular parts of the cytoplasm can be separated from the liquid part of the cell. The red cells intact have a definite respiratory power which after hemolysis is located in the nuclear parts. If the nuclei are well disintegrated by thorough grinding they lose the power of absorbing oxygen, all of which tends to support the theory already based on microchemical and histological evidence, that the nuclei and cell granules are the seat of the oxidation reactions of the cell. Warburg tried to imitate the cell respiratory process by using blood charcoal as the finely divided surface on which oxidation reactions could occur. He succeeded in oxidizing amino acids to carbon dioxide and ammonia by shaking the amino acid solution in air with blood charcoal. The amino acid was adsorbed on the surface of the charcoal and the decomposition was catalysed by some ingredient of the charcoal. He experimented with pure charcoal and with blood charcoal which had been extracted with hydrochloric acid and found these samples to have very little oxidative effect. The addition of certain compounds of iron to these inactive charcoals made them active, after they had been heated to glowing. The effect of cyanides on these charcoal mixtures was similar to that on tissue respiration. The presence of narcotics also inhibited the oxygen absorbing power. Since all of the charcoals used had the power of absorbing

the amino acids it was apparent that the oxidation was dependent upon some other factor in the charcoal. The reactive charcoals tested by Waring all contained iron and nitrogenous material. Charcoals poor in reactivity could be made active by heating with iron salts. Furthermore the cyanides inhibited the mechanism in the charcoal, and Warburg concluded from all of this evidence that the amino acids were adsorbed on the charcoal and then oxidised by means of iron in some sort of complex with the nitrogenous material of the charcoal.

Of the principle combustibles of the cell only amino acids were oxidised by blood charcoal. Glucose and fatty acids were not decomposed. The combustion of glucose and of fatty acids in tissues is retarded by prussic acid, which would seem to suggest the activity of iron in the tissue mechanism. Warburg succeeded in oxidising fructose in a sodium phosphate solution, using iron as the catalyst, and as the reaction did not occur in other salt solutions, it seemed possible that the iron in this instance catalysed the oxidation of a fructose-phosphate derivative. In the oxidations of fatty acids the combination of iron with substances containing the sulphydryl group was significant. Meyerhof and Hopkins oxidised lecithin and linolenic acid and in each case the catalyst was a complex of iron with a sulphydryl compound.

The oxidation of amino acids was accomplished by means of a complex of iron with nitrogenous material. Fructose oxidation was catalysed by means of iron in the presence of phosphates, and the most reactive combination of iron, namely, that with cysteine or glutathione, effected the oxidation of lecithin and of fatty acids with a velocity ten times as great as that in the oxidation of amino acids or of fructose.

The oxidations of amino acids, of fatty acids and of sugars in the tissues are accelerated by complex catalytic agents, and these vary in their composition. Each mechanism contains iron in some form, and to the iron is attributed the power of activating the oxygen and of transferring it to the metabolites to be oxidised. Why iron in one combination reacts quickly and in another slowly, cannot be explained any more than the velocity of any chemical reaction can be explained. In the field of biological oxidations there are many reactions apparently independent of the activity of iron, but the tissue oxidations as investigated by Meyerhof and Warburg are catalysed by means of iron complexes in which other metals cannot be successfully substituted, and iron maintains the central position as the oxygen carrying component of the respiration catalyst.

The Post Operative Treatment of Abdominal Cases.

J. A. MACFARLANE, F.R.C.S., EDIN.

IN the field of surgery it is generally conceded that certain factors may definitely contribute to the success of any operation, or on the other hand may modify the degree of that success. Under such factors one thinks of the general health of the patient, the preoperative preparation, both local and general, the skill and efficiency of the surgeon, the adequacy of his technique and assistance, and finally the post-operative care. In abdominal surgery particularly, this latter factor is of prime importance, and the surgeon who is desirous of attaining the greatest ultimate success can never be unmindful of this fact. In any well-ordered hospital service or clinic, post-operative treatment for the ordinary case becomes a matter of routine, and to the student perhaps such treatment seems of minor importance. Frequently he retains that idea until he has the responsibility of post-operative care thrown upon him either as a house-surgeon or in some similar capacity. With this in view, it may be of value to consider in brief the general care of a case following operation.

General Care.

One cannot overestimate the importance of skilled and efficient nursing. A careful clinical chart should be maintained for thirty-six hours, and longer in the more serious

cases. On such a chart, the amount of fluid intake and excretion should be tabulated carefully and totalled at the end of each twelve hours. If vomiting occurs, the time and the amount should be noted. Periods of sleep and attacks of pain, etc., are likewise noted, so that the surgeon is able to judge of the patient's progress on looking over the chart of this twelve-hour period.

The Fowler position as originally devised for cases of peritonitis, is the most satisfactory for all abdominal cases in the period immediately following operation. As well as affording the greatest degree of comfort, there is probably a lessened possibility of the development of respiratory infections.

Rest is all essential and of the various drugs at one's disposal, morphia is the most adequate in the first thirty-six hours. There are few if any contra indications to its administration in amount sufficient to ensure the necessary rest in that period. The amount of the doses and the frequency should not, however, be left to the discretion of the nurse. Patients vary greatly in their need for sedative. Repeated calls for it demand an examination by the house-surgeon and his satisfaction that the need is genuine. Where the abdomen has been closed without drainage the dressing may be left for five days, unless there is evidence of probable infection in the way of increased temperature and pain. If there is evidence of deep infection in the wound, in the majority of cases it is wiser to apply heat until the collection of pus is superficial enough to be drained by the insertion of a pair of dressing forceps. Haemotomata in the wound should be evacuated on their recognition. Where the abdomen has been drained, the dressing, of course, should be done as often as is necessary, depending on amount and character of discharge.

Common Complications.

Retention of Urine.—In the majority of cases it is unnecessary to resort to catheterization. Repeated applications of heat to the lower abdomen, fluids in quantity, and if these fail, the administration of a small enema will frequently give the desired result. A catheter at any rate should not be used until simpler measures have failed, and not until one is satisfied that the patient is really uncomfortable. The eight-hour period so frequently set down, can be safely passed in many cases without any evil results. If catheterization must be done, it should be done with strictest care in regard to technique.

Vomiting.

In some individuals post-anesthetic vomiting is particularly obstinate and may interfere with the administration of the necessary fluids by mouth for some little time. Vomiting occurring in the second twenty-four hours, aside from protracted post-anaesthetic nausea, is of more serious importance. It may be due to continued peritoneal irritation, inflammatory in origin; it may be partly due to general abdominal distension, or it may be due to acute gastric dilatation. In post-anaesthetic nausea, one resorts to the milder measures. Mustard paste to the epigastrium frequently gives relief. The varying of fluids given, soda water is tolerated where other fluids have caused nausea. Milk of Magnesia, Bicarbonate of Soda, Aqua Menth Pip., these should all be tried before one resorts to gastric lavage. When they have failed, particularly in neurotic patients, one introduction of the stomach tube is frequently sufficient to relieve them permanently. In the other forms of vomiting it is necessary as a rule to cut off all fluids by mouth, and resort to frequent use of gastric lavage. In cases of general abdominal distension or ileus the vomiting is only part of a more serious condition and must be treated as such.

Acute dilation of the stomach is a very serious and difficult post-operative emergency. It occurs after the first twenty-four hours, is characterized by the effortless vomiting of great quantities of greenish fluid, at very frequent intervals, the rapid dehydration and ultimate death of the patient if left untreated. In these cases, one must first attempt to keep the stomach empty, and secondly, restore the fluid loss to some degree at least. Lavage should be done at two-hour intervals; in the period intervening the patient should be rested in the prone position and the foot of the bed slightly elevated. Fluids will be best given

by the intravenous route either normal saline of 5 per cent. glucose in a solution of normal saline.

Distension.

No other symptoms give rise to so much discomfort in the post-operative abdominal case. There is much variation in opinion on the time that laxatives should be given, following an operation in which the abdomen has been opened.

In a paper that has to do with general treatment of varied types of abdominal cases very little can be said in that regard. However, one is inclined to the opinion that the average case is better served by being kept comfortable with Enemata until he can take Liquid Parrafin by mouth, and that this be given in sufficient quantity for the remainder of his convalescence to ensure the necessary bowel movements. The more drastic purgatives, particularly calomel, are unnecessary, and give rise to much discomfort. For the relief of post-operative distension we can do much. Heat in the way of linseed poultices of generous size frequently applied, gives a marked degree of comfort and relief in the early stages. It should be used in those cases which are likely to become distended, immediately the patient is recovered from the anaesthetic. An enema may be safely given after twelve hours. It is seldom necessary to give it before that time. There are varying opinions as to the efficacy of the different enemata. A simple enema is usually given in the early stages; if this is ineffectual, then a turpentine enema. A milk and molasses enema is frequently of value where others fail. If the patient is tired out, a rectal tube will often give relief.

The drugs at one's disposal are Eserin and Pituitrin. Eserin is best given in 1/50 gr. dose, four doses by hypodermic injection at three-hour intervals and then discontinued. Pituitrin is given in ½-1c.c. doses hypodermically, and is usually followed by administration of a turpentine enema. These drugs may also be given together, Eserin gr. 1/100 and Pituitrin 1c.c., and this followed by a turpentine enema. This frequently is effectual where other measures have failed.

Ileus.

Persistent abdominal distension and the failure to pass gas by rectum is due to either a mechanical obstruction or to a paralytic condition of the intestine. The latter condition is frequently encountered in the post-operative course of acute inflammatory intra-abdominal conditions such as peritonitis, pancreatitis, diverticulitis and in perforated gastric and duodenal ulcers. Its treatment presents the most difficult problem in post-operative care. Persistent distension is accompanied by vomiting and the patient rapidly becomes dehydrated and weakened. The replenishment of the body fluids is all important in the treatment. Much has been written on the efficacy of administration of salt solution of high percentage in these cases. In many cases an investigation of the chemistry of the blood reveals a markedly lowered chloride content as well as a lowered carbon dioxide combining power, and with this in view salt solution of 2 per cent., 6 per cent. and 20 per cent. concentration have been given in order to restore that content. Ammonium chloride in 100 grain doses by rectum is also given to increase chloride content. However, one must not lose sight of the fact that the crying need of these patients is fluids. At least 100 ounces of fluid in each twenty-four hours should be given to any adult patient following a serious abdominal operation. In cases of Ileus this may be increased very considerably. It is usually necessary to supplement that given by interstitial and rectal methods by daily intravenous administration. As much as 800 c.c. may be given to an adult by the vein at one time if given fairly slowly. About one ounce per minute is a reasonable rate. Apparatus may be arranged with little trouble so that fluids may be given by a drop method into the vein and the administration continued almost indefinitely. The accompanying nausea and vomiting is treated by gastric lavage as often as necessary and every effort is made to relieve the distension by the measures mentioned above. With all, one should not forget the need for rest. Where intravenous therapy is being used, enemata being given repeatedly, and the various other measures employed for the relief of the con-

dition, the factor of rest is frequently forgotten. There should be periods of an hour or two at least when the patient should be kept free of treatment, and if necessary given an opiate to induce sleep.

Such cases require the unceasing care of the house-surgeon and nurse in charge and the careful judgment of the surgeon. It is not always easy to determine whether such a case may be developing a mechanical obstruction, a very real possibility in such cases as general peritionitis. Mechanical obstruction is, of course, an indication for immediate operative interference and in a certain number of obstinate cases of paralytic ileus, one must also consider the question of a jejunostomy. The drainage of the distended jejunum may be just sufficient to turn the scale and enable the patient to overcome the toxaemia which has resulted from it.

A Review of the Etiology of Puerperal Infection.

P. J. MURRAY, '27.

PUERPERIUM is the interval or period following the termination of labour during which the uterus returns to its normal non-pregnant state.

Puerperal infection refers to all the various moribund conditions which result from the entrance of infective micro-organisms into the female generative tract during labour or the puerperium.

Puerperal infection, puerperal fever, puerperal septicaemia and puerperal sepsis are often used as synonymous terms, but the one of choice is puerperal infection.

The older term, puerperal fever, is unsatisfactory in that it suggests the essentiality of the affection—further, it emphasizes the febrile phenomena and does not stress its infectious nature. The terms puerperal sepsis and puerperal septicaemia represent degrees of the severity of the infection and are not satisfactory because in many instances the infection results in perfectly localized inflammatory processes to which such terms cannot be applied without violating the established rules of diction.

History.

That puerperal fever existed among primitive peoples may be seen from a study of the Ayur Veda of Susruta, a thousand years before Christ. Hippocrates in 400 B.C. described cases so well that they could be placed in the modern text-book. The means of prevention used by the Ancients varied with the peoples—isolation of the parturient, the cleansing bath in the stream after labour, the fumigation of the vulva with aromatic herbs and fumigation of the apartment. The disease is mentioned by Celsus and Galen. The first recorded epidemic occurred at Leipsig in 1652-1655, and was reported by Heroieux. The great French obstetrician, Mariceu, ,reported an epidemic in 1660 in the Hotel Dieu, with a mortality of 67%. In 1831 the mortality in Paris Maternite was nine per cent.. It raged in Vienna where the great Rokitansky held his clinic, and England was by no means free of the scourge.

At that time the theory of the disease most accepted had been advanced by Hippocrates, namely, that the disease was caused by the suppression of the lochia.

Puzos of Paris taught the "milk theory." He contended that during pregnancy milk secretion begins, but it is directed to the uterus where the fetus uses it for nourishment. After labour it is excreted by the breasts. If for some reason it is checked, so-called milk metastases occur and cause fever. There the milk appears in the lochia (purulent lochia of sepsis), in the peritoneal cavity (the pus of peritonitis), in the pleural cavities (empyema) and in the joints (pyaemic arthritis).

In 1843 Oliver Holmes demonstrated the infectious nature of the disease in a convincing style by an array of clinical reports—pointing out that it was carried from one patient to another as a contagion, but his views drew such a storm of abuse that to avoid conflict, he dropped the issue.

Fortunately for womanhood a brilliant champion arose to fight their battle in the

person of Ignaz Philipp Semmelweis, a young assistant in the obstetrical clinic of Vienna. The ward he was on bore an unenviable reputation in that the mortality was terribly high, while the clinic conducted by the midwives was much lower. He noted that the disease prevailed with irregular, unassignable fluctuation year in and year out in the hospital, but not in the homes; that women who were delivered on the way to the hospital seldom sickened; that parturients who had long instrumental deliveries were nearly always infected; that maternities where students were not taught showed lower mortality; that overcrowding had no influence; that fright and nervous influence had no effect; that the disease affected the married as well as the single; that often the new-born died of the same disease as the mother. Following the death of a personal friend, who some days previous had pricked his finger while doing an autopsy and whose body showed similar pathological changes as those of the unfortunate puerperas, he decreed that all vaginal examinations must be preceded by washing the hands in a solusion of chloride of lime. The mortality fell almost immediately to 1.27%, even lower than it was in the midwives' clinic. In consequence of this observation he advanced the following theory: "Puerperal fever is a resorption fever brought about by the introduction of decomposed animal matter into the genitals. This matter could come from cadavers, from infected wounds of all kinds, and was carried into the genital tract by the fingers of the accoucheur, the instruments, the douche-nozzle, the sponges, the infected air, the bed linen, the bed pans, indeed everything that could transport the decomposed animal matter from its source to the parturient canal."

It was not, however, until the researches of Pasteur and Lister had resulted in the discovery of organisms in infection that the views of Semmelweis became generally accepted.

In 1864 Rokitansky discovered the presence of organisms in the lochial discharge of women suffering from puerperal fever, and in 1865 Meyerhofer was able to identify the streptococcus. In 1869 Coze and Feltz were also able to detect organisms in the blood in a fatal case of puerperal infection. It was not, however, until the year 1879 that Pasteur was able to completely establish the relation of these organisms to puerperal infection. These observations were speedily confirmed by Koch, Doleris, Fraenkel, Doderlein, Kronig and Menge, and others, and form the basis of our present-day knowledge of the bacteriology of puerperal infection.

Micro-organisms of Puerperal Infection.

A large number of micro-organisms have been discovered in cases of puerperal infection, and they may be conveniently divided into these groups:

1. Pyogenic bacteria. These are the organisms usually found in surgical wound infection. The streptococcus hemolyticus is by far the most frequent cause. Not rarely the staphylococcus, B coli or B pyocyaneus are present.

2. Organisms of certain specific diseases. The most important of these is gonococcus, which grows abundantly in the vaginal secretion. The pneumococcus, the diphtheria bacillus, the bacillus typhosus has also been found in rare incidents.

3. Anaerobic putrefactive bacteria. These organisms are very numerous and only develop freely in the presence of retained secretion or injured tissue.

Of all these organisms by far the commonest one is streptococcus hemolyticus—it being held responsible in at least 80% of the cases, and it has been proved beyond question that this organism is capable of giving rise to all the varied clinical types of puerperal infection—that is to say it is the organism present in a large proportion of mild cases and in almost all the severe forms of the disease.

Conditions Predisposing to Infection.

1. Condition of the individual.

The general health undoubtedly exerts an influence on the predisposition to the occurrence of infection. Depreciation of vitality by overwork; insufficient food; alcoholism; and bad sanitary conditions probably exert their influence by impairing the bacteriolytic and phagocytic power of the blood and tissues, thus diminishing the in-

tensity of the local reaction and facilitating generalization of infection.

2. Condition of the generative tract after delivery.

During pregnancy the uterine cavity is closed by a firm plug of cervical mucus which effectually prevents the invasion of organisms.

The flow of blood, the flushing action of the liquor amnii, passage of the child's body with progression and recession of the foetal head and the herniating of the placenta through the membranes during labour, tend to cleanse the vagina, while the lochia by its bactericidal action attempts to destroy bacteria. The cervix is widely dilated—it has just undergone a great amount of stretching, the result of which is laceration in some cases, and even where it is not evident, such a strain on the tissues that the local resistance is lowered.

The interior of the uterus is reddened, soft and pulpy, with blood clots and debris remaining on the ragged placental site—an ideal soil for the growth, multiplication and spread of organisms.

3. Character of Labour.

Although infection may arise after the most straightforward delivery, its occurrence is much facilitated by premature rupture of the membranes, prolonged labour, and obstructed labour.

4. Presence of organic material in the generative passages.

5. Presence of a wound.

Modes of infection:

1. Heterogenetic.
2. Autogenetic.
3. Haematogenic.

Heterogenetic.

By this is meant the introduction of organisms from without. This is by far the most frequent and important mode of infection. In some instances the organisms may be derived from organisms existing in or on the patient herself, because of the close relation of the rectum, which teems in bacteria, and also the skin of the healthy individual possesses an abundant bacterial flora, but in most cases they are derived from sources external to the patient herself and are introduced by the hands of the obstetrician. Liability to infection is in direct proportion to the frequency of examination and is markedly increased by manipulative procedures and operative deliveries.

Autogenetic.

This term is restricted to those infections which arise from organisms existing in the genital tract of apparently healthy women during normal pregnancy and labour. Much difference of opinion has existed on the possibility of infection from from this cause and for the solution of the problem it is, in the first place, essential to ascertain if the genital canal normally contains organisms capable of producing infection. This has been the subject for much research. Doderlein has shown that during pregnancy there is a decrease in the acidity of the vaginal secretion and that this permits the growth of bacteria which in the non-pregnant are destroyed; however, the evidence at the present time goes to show that hemolytic streptococci, the commonest cause, are not found, and it is very unlikely that the non-haemolytic streptococci found can become haemolytic.

Haematogenic.

This means the conveyance of organisms from an infective focus in some other part of the body to the genital tract by the blood stream. The possibility of this occurrence has been experimentally proven by Orthmann and Hillendabl, using subcutaneous injections of pneumococci in pregnant guinea pigs, and clinical evidence has been afforded in a few cases—especially the pneumococcus.

Very recently it has been suggested that that type of puerperal infection which rapidly becomes a septicaemia may be due to a specific strain of streptococcus. Clinical evidence in support of this idea is manifested by the fact that highly fatal epidemics have broken out in the most carefully conducted maternity hospitals which have been extremely difficult to control. As yet it has not been definitely established, but it is interesting to note that following on the dis-

covery that streptococcus of scarlet fever produces a true exotoxin, attempts have been made to isolate an exotoxin from this strain of streptococcus with some measure of success, and it is possible that in the future an effective serum may be produced.

Summary.

1. Puerperal infection is wound infection resulting from the introduction of pyogenic organisms into the generative tract either before, during or immediately after labour.

2. The offending organism in practically all cases is streptococcus of the haemolytic group.

3. The mode of infection is mainly exogenous—the organisms being carried in by the hands of the obstetrician or instruments.

4. The means of prevention are obviously:

(1) To make as few vaginal examinations as possible.

(2) To make all vaginal examinations with surgical means of precaution.

(3) To refrain from the use of all unnecessary measures which will tend to increase the probability of laceration.

Focal Infection and Elective Localization of Bacteria.

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THE title chosen for this short paper embraces two views of infection which have caused much discussion. I will endeavour to give as concisely as possible some of the leading features of the different points of view, especially on elective localization since the significance of local infection is generally accepted.

A "focus of infection" may be defined as a circumscribed area of tissue infected with pathogenic micro-organisms. The focus may be primary or secondary. The primary focus is usually in tissues communicating with a mucous or cutaneous surface, but not necessarily so. The very great importance of foci of infection is recognized by all, and a vast amount of study has been devoted to the many problems arising from the primary and secondary results of such infections. In the broader use of the term "focus of infection," there must needs be included practically every microbial infection which starts in the tissues at any portal of entry, where it becomes established, and whence it spreads to other tissues. All surgical sepsis is of this type, as are all of our common infectious diseases. The term, however, has become, in the minds of most practitioners and investigators quite definitely limited to a particular kind of local infection in which there is relatively little evidence of local activity; a chronic or low grade infection, often extending over years, and frequently discovered not from the local symptoms, but because of the secondary foci in other organs and tissues; a partial adaptation of the local tissues to the parasite, a kind of symbiosis analogous in some ways to the "carrier" state. The sites of these primary foci are widespread and include: infected areas of teeth and jaws, with pyorrhoea dentalis and alveolar abscess; tonsils (faucial and nasopharyngeal); mastoid, maxillary, frontal and other accessory sinuses; cutaneous, subcutaneous and submucous abscesses (osteomyelitis, for example, is almost exclusively associated with some form of skin infection); bronchi and lungs in chronic infection; gastro-intestinal tract and auxiliary organs of digestion, as the gall-bladder and appendix; intestinal ulcers and the bowel in stasis; the genito-urinary tract of male and female, and in fact in almost any region of the body.

The secondary infections which attract the attention of the patients and because of which they seek medical aid, are as widespread as are the primary ones, and many of the infections mentioned for the primary sites may be in one case primary and in another secondary. The serious conditions which are known to follow focal infections are numerous, such as bacterial endocarditis, myocarditis, iritis, arthritis and many more.

The more intimate knowledge of the

mechanism by which bacteria cause these secondary infections is of importance from many points of view, but particularly from that of prevention. One of the explanations for the phenomena of secondary infections is that given by Rosenow in his "Elective Localization" theory, supported by Billings of Chicago and other clinicians, in fact "it has influenced clinical thinking to an extent not warranted by experimental fact." Zinsser considered it a dangerous thought to spread broadcast and I agree with him. It was hoped the theory would have died a natural death, but it has not done so, and I have undertaken in this short article to indicate some of the reasons which make it untenable, and indeed dangerous.

First, just what is it Rosenow means by "Elective Localization?" He believed, and still believes, that certain bacteria (particularly certain non-hemolytic streptococci) acquire specific affinities for particular tissues. This means that, for example, a streptococcus which has caused an endocardial lesion in man, if it is injected intravenously into another animal (e.k., the rabbit) or gains entrance into another human being, will tend to locate in the endocardium and cause an endocardial lesion. This tendency is supposed to be very sensitive, being rapidly lost on artificial media, and as a result of animal passage. Rosenow has endeavoured to demonstrate this quality of streptococci for lesions of the appendix; stomach and duodenum; gall bladder; endocardium; joints; myocardium; skeletal muscles; kidneys, as in nephritis and nephrolithiasis; the nervous system, as in herpes zoster; spasmodic torticollos, epidemic hiccough, lethargic encephalitis; skin in erythema nodosum; eye in iritis, and other tissues. The functional activity of the analogous organs or tissues in the animal must often be quite different from that in man as in the appendix and the skin of the rabbit.

The germ of truth which has helped to make this theory acceptable is to be found in the superficial reading of certain other diseases. Tuberculosis would appear to have a selective localization in the lung of man, but the localization in animals—rabbits and guinea pigs—depends on the anatomical differences in these animals and not upon the organism. Tubercle bacilli from infected joints, kidneys, meninges, do not show any tendency to locate in these organs when injected intravenously into animals. The criterion of comparison in tuberculosis is the type of lesion and not its locality. Meningococcus has a tendency to locate in the meninges in epidemic meningitis but not so in animals, and there is an increasing number of cases of meningococcemia with other localizations in man. Nevertheless, there is no question of a certain kind of "elective localization" for certain diseases, since this is what really differentiates many of our specific diseases, as typhoid fever, cholera, diphtheria, and many others. In all these examples there is a mutual relationship between the bacteria and the tissues involved, but in Rosenow's theory the dominant character is to be found in the members of the streptococcus group, and it manifests itself practically independently of the local conditions. There is just enough mystery about this theory to make it intriguing, and it throws the responsibility on chance rather than on the condition of the patient. If you happen to get a streptococcus in your tonsil, tooth or sinus, which in some strange way (increase or decrease in virulence, adaptation to varying oxygen tensions) has taken on qualities of specific affinity for the appendix, then the chances are that you will develop an infection of the appendix. Well, what are the facts? It will be granted that, armed with such a theory, the experimenter, being human, would tend to try to confirm it. If the streptococci from a case of iritis had caused in the animal of experiment, lesions in the eye, appendix and joints, there would be a tendency to overemphasize the lesion in the eye.

In carefully analyzing the experiments of Rosenow, it is to be noted that there are practically no constants. These are essential before we can compare results in experiments of this type. The amounts and age of culture injected, the age and species of animals, the number of injections, the time between injections, the seasons of the year, and many other factors all varied. The strains of streptococci used were rarely obtained from the actual tissue-lesion, but usually from the "apparent atrium of infec-

tion," and they were usually injected without any attempt to obtain pure cultures, because the "affinity" was easily lost on media. Many of the strains are only ex post facto specific strains. No mention is made of "spontaneous" infection in any of his hundreds of animals, and no difference is made in interpreting results between animals dying or those chloroformed or otherwise killed. Even if we granted that all these variations would not vitiate the results, it is found that Rosenow's own figures and results do not indicate any greater tendency on the part of the "specific" strains to locate in the analogous tissue than would be expected on the law of chance. The method of using his statistics gives the impression that such a tendency was present in his cultures, and indeed it has so been interpreted by those supporting this theory, but using his tabulated results in what we believe is a more logical way, this tendency disappears.

I have estimated from his percentages the actual number of animals showing lesions in a particular tissue, and of these positive experimental results, have determined the percentage due to specific and non-specific strains. The results showed no important constant difference. For example, 130 animals showed lesions with haemorrhages in the stomach and duodenum, and of these 48 per cent. were "ulcer of stomach strains," and 52 per cent. other strains; 92 animals showed ulcers in stomach and duodenum, 67 per cent. with "specific" strains, and 33 per cent. with other strains; 76 had lesions in the gall bladder, 43 per cent. being from "specific" strains and 57 per cent. from other strains; joint lesions were found in 131 animals, "rheumatic fever" strains giving 36 per cent., and other strains 64 per cent.; appendix lesions occurred in 65 animals, and of these, 71 per cent. were after "specific," and 29 per cent. after other strains; lesions in the endocardium were noted in 122 animals, "endocarditis" strains accounting for 30 per cent., "rheumatic fever" strains for 27 per cent., and other strains for 42 per cent.

In other words, there is no evidence in the results on which the theory is based, that "elective localization" as defined by Rosenow, plays any role in these infections. Rosenow's results, however, are of great importance in indicating that streptococci can locate and cause lesions in a great variety of tissues, and his work is most valuable for this reason. It is with the interpretation of his results that I disagree.

The experimental method of injecting animals intravenously with large doses is probably not very close to what actually occurs in natural infection, and I will try to give briefly my view as to the mechanism involved in this type of infection as it occurs in man. The streptococcus viridans group of bacteria is most often involved, because it has certain biologic characters. These streptococci are highly invasive, that is, they pass the normal barriers of bodily defence relatively easily. There is usually no marked clinical evidence of their presence, little or no rise of temperature, but the patient may have a general feeling of lassitude—the neurasthenic feeling—and the leucocyte count may be altered. They are not dramatic fighters, these streptococci, and are comparatively easily overcome if there occurs a sharp reaction on the part of the body. In this they differ greatly from their relatives of the hemolytic group. They are like snipers watching their opportunity to strike from their focus of infection, and they send out foray after foray, increasing the damage they do at each attack. These infections are dangerous because we do not recognize them. Patients consult medical men only after numerous attacks and because of some disturbing symptoms. It is probably the most difficult problem in the field of medicine to control and understand these sub-clinical infections.

There are two main requisites before such infections occur. A focus of infection of the type indicated above, and a condition of lowered resistance in the tissue to be involved. The "condition of lowered resistance" in the local tissue is just as big a problem as the "focus of infection." These infections are very frequently carried in the blood stream and must be arrested in the tissue and remain there long enough to multiply before the tissue is actually infected. It is, of course, not necessary to postulate blood stream transmission in all these infections, but this probably is the most frequent

occurrence. The factors, therefore, must be considered which favor the liberation of the bacteria from the focus, and those helping their stoppage in the tissues. The conditions found in the capillaries are of the greatest importance in determining what will take place in either case and the various influences affecting the capillaries are therefore fundamental in such a study. The capillary supply differs in different tissues and organs; the spleen, for example, is like a great capillary organ. The capillaries and the terminal arterioles and venules regulate the oxidation processes and the food supply of tissues, and are controlled locally by local reflexes, as well as by general reflexes. The endothelial lining varies in its permeability and is sensitive to many substances in the blood, such as hormones, toxins, and changes in the hydrogenion concentration. When an organ is actively functioning, the capillaries are more or less fully dilated, and the blood flow is rapid. During general bodily exercise, for example, we know that the capillaries in the muscles are thus functioning, and the same is true in the mucous membranes and skin vessels. The capillaries in a focus of infection join in the general response and bacteria are more readily carried from such a focus into the general circulation under these conditions. The chances of stoppages in the capillaries are, however, at this time minimal unless other factors intervene. The period of danger is after the exercise has stopped, when the capillaries automatically close down in response to a lessened function. The athlete trainer recognizes this danger and efforts are made to bring about a gradual return to normal, to wrap up and keep warm after exercise. A prolonged chill at such a time causes a sharp contraction of skin capillaries, followed by those in the mucous membranes and in certain organs. This is the time when bacteria, if they have been in the blood, are liable to be stopped in the tissues and cause a local infection. Such an infection may be rapidly overcome, or it may do damage in the tissues of the capillary bed, and though eventually overcome, may have left tissue changes sufficient to interfere with capillary response on another similar occasion. Such infections, moreover, are most probable in tissues which have sustained a previous injury by physical or other means. This is particularly noticed in rheumatism and osteomyelitis.

Overworking the function of an organ, be it kidney, heart, pancreas, or any other organ or tissue, is liable to lead to fatigue of its capillary mechanism, and open the road to infection. Most of these infections are passing affairs and unnoticed, but after frequent repetition they rise to the surface of clinical observation. There are many other factors besides capillary contraction or stasis which may favor local infection, such as: increased viscosity of the blood following loss of water through perspiration; increased local content of carbohydrate from diet, lowered tolerance or as a result of stasis; food or other anaphylactic reactions rendering the capillaries more permeable and leading to undoubted local damage even without infection; changes in hydrogen-ion concentration; and many other conditions which alter the local response.

That non-hemolytic streptococci are not specific even as a group for these infections, is shown by the occurrence of similar infections with other types of bacteria, as the gonococcus, micrococcus tetragenous, staphylococci, hemolytic streptococci and other types. Nevertheless, the streptococcus viridans group being found most frequently in the various foci of infection, producing as it does a primary low grade type of infection, and because of its high invasive power, should be considered as the most important group of organisms concerned.

Realization of the potential danger of foci of infections and a more complete knowledge of the factors affecting capillary response, and the conditions favouring local injury and infection, may lead to at least a better understanding of the processes involved, which in turn will be an aid to treatment. There is no need to postulate elective affinity on the part of the bacteria when we have such a weight of evidence indicating the determining importance of local conditions in the tissues. The theory of "Elective Localization" 'is not only unnecessary, but is dangerous, since it leads to a belief in the hopelessness of prevention, and de-

ludes the physician into thinking of the mystery of the bacteria, instead of the local and general conditions which can often be remedied or alleviated. I do not wish to underestimate the importance of foci of infection and the urgent need of removing these when found, but I do wish to stress that there are many factors involved and that advance cannot be made by adding mystery to ignorance.

The Place of Anatomy in Medicine.

SIR ARTHUR KEITH, F.R.S., F.R.C.S.

THE life of a medical student, especially in its earlier years, is full of novel experiences and strange adventures. There is, for example, the October morning when he shyly pushes open the dissecting room door and, putting on an outward air of bravado to cover his inward trepidation, advances in search of the subject which is to afford him his first practical experience of human anatomy. As he discovers the object of his search we see him don immaculate apron and sleeves, undo the lid of a brand new case of knives and seating himself by the subject, open his dissecting manual at the section devoted to the arm, impatient to commence operations. As he makes these preparations a sense of deep satisfaction takes possession of him, for he feels that at last he is to begin the real study of his profession. He had listened, but with only a half-hearted acquiescence, when his lecturers on chemistry, physics, and biology had claimed that theirs were the basal subjects of scientific medicine. But now all doubts had vanished; the stark subject which lies stretched on the table beside him measures the limits of his life's work; it is the field of all medical and surgical endeavour. No wonder, then, that the young student, as he makes his first incision and meets with those mysteries spoken of as superficial and deep fascias, feels that at last he has entered his profession. It was thus, in October days long gone by, that young medical students named William Harvey, Thomas Sydenham, John Hunter, Edward Jenner, Charles Bell, Marshall Hall, James Paget, and Joseph Lister entered the profession of medicine. Their first incision was also their blood-baptism to the profession of medicine. And so it will be in the future. Scalpel and forceps can never become antiquated, for as long as medicine endures, its practitioners must learn, by their use, the structure and working of the machine they would mend.

It is difficult for us who have grown grey in the service of medicine to recapture the enthusiasm, resolution, and outlook with which we first entered the dissecting room. The task ahead seemed so simple; the kingdom we had to conquer, the human body, had but a few parts—a head and neck, thorax, abdomen and pelvis, upper and lower extremities, and some special organs of sense. Its bones and muscles had been duly numbered and named; the shape and surfaces of its organs fully described; all its vessels and nerves had been charted. With the optimism of youth we felt certain that before another October came around we should be masters of anatomy; by that time we hoped to have built up in our minds a picture or lay figure of the human body. This lay figure, reared in the dissecting room, we proposed to carry with us to the physiological theatre and laboratory, and there, as it were, have its machinery set in motion. It was to be for us what Maconochie became for Sir James Barrie—an alter ego which was to keep us company every day and everywhere. This anatomical Maconochie was, in due season, to go with us to the pathological department, and there his inward parts were to suffer all the diseases to which human flesh is liable; when the time came all the remedies of the pharmacopoeia should be tested on him and all his systems marked with therapeutic tags. He was to accompany us to the wards—if we ever got so far—there to serve as a lay figure on which we should graft experience gleaned from surgical and medical cases. Nay, in the distant future, when we had become practitioners, we conceived this lay

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figure, first built up by scalpel and forceps in the dissecting room, as the companion of our rounds—an epitomized embodiment of our medical knowledge—a representation of our whole stock-in-trade.

Why is it that in so many of us the zeal and enthusiasm with which we entered the dissecting room fade so soon and the anatomical "Maconochie" of our dreams is dead before the morning dawns? The art of dissection is laborious; the accounts given in text-books are accurate, detailed, and prolix, but as dead as the parts described. Very soon, unless a teacher is at hand, the youthful student of anatomy becomes submerged in a chaos of fact. Some of the best minds which enter the profession of medicine turn from anatomy with contempt; for them it is but a catalogue of names. Besides, the student who would realize his early ideal and the teacher who would willingly help him, have to keep an eye on examination papers. In every question the candidate is desired to "describe" or "enumerate" or "give an account of" shapes, relationships, and attachments of certain structures. All references to the purposes these structures serve in the living body are excluded. The student, in such circumstances, abandons his earlier and truer ideal and accepts the conditions which tradition has made for him.

British teachers of human anatomy have realized more fully than those of any other country what is wrong with the subject. This subject has been killed by tradition, and if it is to take its place as the fundamental subject of medicine, they have to make it alive again. They are now revivifying it in many ways—by using living models as the subjects of their demonstrations and lectures, and by applying the discovery of Rontgen to the study of the living body. They desire that their students should carry from the dissecting room, not a memory of dead subjects, but of live men and women. No matter how high the ideals of the professor may be or how actively the demonstrator may supervise dissections, if this aim is to be attained—if the dead subject is to be made alive—each student has to do it for himself or herself. No one can perform this miracle for them. It is an easy matter to reflect skin, separate parts, and give a neat and clean demonstration of structures; to memorize descriptions is, at most, only a mechanical art, but to build up a living picture of each part as it is dissected, to ask and answer the questions: What is the use of this structure? Why is it shaped so? entails real mental labour. Without this travail of the mind the labours of the dissector are thrown away and his time is largely wasted. It is this latter aspect of his work in the dissecting room which the modern student of anatomy fails to appreciate, and towards which his best books give him so little guidance. He clings to the hope that there is some easier way—some short cut—some system of mnemonics which will resolve his difficulties and assist him to satisfy the demands of his examiners. Sooner or later he will discover that in anatomy, as in all other branches of knowledge, there is for him but one royal road to learning—a diligent and intelligent application of all his mental powers.

It does not seem natural that the study of function should be separated from structure in the dissecting room, and it may happen that some student who reads this article will care to find out how anatomy became divorced from physiology. If he turns to the writings of John Hunter, which cover the latter half of the eighteenth century, he will find that anatomy, physiology, and surgery were taught by one man. John Hunter was a splendid anatomist, but when he dissected, his aim was to find out, not what an organ looked like, but what it did. He studied anatomy and physiology to help in diagnosing and treating his patients. He studied dead things to understand the living, and observed living things to understand the dead. Then if the inquirer wishes to know how anatomy and physiology were taught early in the nineteenth century he should consult a text-book which was bought and read by medical students one hundred years ago. On its title-page this text-book is described as: "The Anatomy and Physiology of the Human Body, by John and Charles Bell (sixth edition, in three volumes, 1826). These two brilliant brothers, like Hunter, taught anatomy and physiology and practised surgery. In their great text-book anatomy and physiology are blended

to their mutual advantage. When the text-books which were used by students of the following decade—the "thirties" of last century—are consulted it will be seen that a "judicial separation" by mutual agreement had been effected between anatomy and physiology. Not a teacher of the time, so far as one can find, entered any form of protest. Indeed, we have only to turn to a book which every student of medicine should read—"Memoirs and Letters of Sir James Paget"—to see that the change received an enthusiastic welcome. The passage which I quote relates to 1842, when Paget was 28 years of age, and just beginning to get a foot on the first rung of the professional ladder:

"The time had come, in fact it had long ago come, when it was necessary to separate the teaching of physiology from that of anatomy. The separation had been made some years before at University College, and at the Aldersgate School, the rival neighbours of St. Bartholomew's, and in some other schools. It was now resolved to make it ours; and my election to the physiological lectureship was sure. The plan was to have a lectureship on descriptive anatomy and another equal in rank and pay on general anatomy and physiology."

In this way anatomy became divided into two branches—descriptive anatomy and general anatomy—physiology being appended to the latter. Very soon descriptive anatomists were named "anatomists" without any qualifying adjective, and general anatomists became known as "physiologists." No doubt the separation of anatomy into two branches had become inevitable, but the student of to-day may wonder why cleavage took the line it did. He may wonder still more how it was that the anatomists of that time were content to hold the husk of the human body when other anatomists ran away with the kernel and called it physiology.

A search for an explanation will carry the inquirer into one of the most interesting episodes in the history of medicine. A reference to the writings of Dr. Robert Knox —a brilliant if somewhat unbalanced exponent of the anatomy of his time—throws much light on the matter. Knox was never tired of extolling the marvels of the new science invented in the schools of France—the "science of descriptive anatomy." The anatomist's sole business, he held, was to give an accurate description of structure. There was also a science of form—"morphology"—but that, he held, had nothing to do with so mean an aspect of matter as function. Probably British anatomists would not have been content to remain mere "describers" had not Sir Richard Owen, during the "forties" of last century, leavened their subject by infusing into it his archetypal speculations. Owen's speculations were dying when, in 1859, Darwin led anatomists away on a new and very interesting chase—the search for the origin of man's body. Then, later in the nineteenth century, came the study of the human embryo, and the light that it threw on man's evolution. More and more the anatomist became the historian of the human body and concerned himself less and less with the action of its parts.

Without doubt anatomy as a subject has prospered and attained enormous proportions since the disruption effected almost a century ago. Its sister subject, physiology, has thriven out of knowledge. But the student of to-day, when he becomes the medical man of to-morrow, and has to relieve the ever-growing congestion in medical education, will doubt if anatomy and physiology have developed along the lines which are most profitable for medicine. For if he surveys the real additions which have been made to human anatomy in recent years he will see that they have been made not by anatomists, but by physiologists. Gaskell, Langley, Ferrier, Sharpey-Schafer, Sherrington, and Head have been the best British anatomists of my time. Indirectly Joseph Lister has done more than all of them for the progress of practical anatomy, for he it was who gave the surgeon access to all parts of the body, and with every departure in surgery there opens up a new field in practical anatomy.

A renaissance is on foot in human anatomy. For the good of medicine and the right progress of their own subject teachers of anatomy are no longer content to confine their efforts to mere description. They feel

that the man who describes the anatomy of the brain and central nervous system must also, if labour is to be saved and time economized, deal with the mode in which its several parts perform their functions. Who could describe the human heart intelligently unless he explains its uses as a pump? Any account of the lungs, thorax, and respiratory passages is useless if the mechanism of the act of breathing is omitted. Sooner or later —and the sooner the better if the student is to be served—a new dividing line has to be drawn between the departments of anatomy and physiology, and that dividing line should not separate structure from function.

I seem to have struck a note of discontent with the present state of anatomy. An impression of this kind is not the one which I wish to convey to young men and women who are now beginning the study of the human body. A knowledge of how this body is made and how it works must provide the basis for all the more practical subjects of medicine. The beginner has to face the fact that to attain such a knowledge requires regular and unremitting application. Application, however, is not enough to attain success; toil soon degenerates into sheer drudgery unless interest is stimulated and maintained. How is this interest to be maintained?

There are many ways. One of the best is to acquire the habit of reading medical journals; never a week passes but there are reported clinical and other cases which have a bearing on anatomy, and in this way students come in touch with the latest applications of their subject to medicine. Besides, this habit of keeping in touch with medical literature is one worth acquiring for its own sake; it will stand students in good stead when they have become busy practitioners or have joined the ranks of consultants or of specialists. Another way of maintaining interest in anatomy is to make occasional visits to the operating theatre, to out-patients, or to the X-ray department. A visit to the post-mortem room will often give a practical bearing to what the student may have observed in the dissecting room. A glimpse now and then at the pages of "Proceedings" and other publications issued by anatomists and physiologists will place him in touch with subjects which are interesting his teachers and reveal the lines along which new fields of research are being opened up. At least a pastime of this kind will remove from the student's mind a delusion widely held by the public at large—that all worth knowing about the human body has been discovered long ago.

I would not take away a single moment of the leisure which the student proposes to give to general literature—nor to amusement or sport—but there are many books on medical biography which will well repay study and help to maintain interest in anatomy and physiology. Especially would I commend the lives of Harvey, Hunter, and Marshall Hall. Every student should get to know John Hilton's book on "Rest and Pain," not only for what he has to say, but also for the way he says it. Then there are a number of excellent modern books which deal with the evolution of the human body, and explain the history of its many curious structures. For my part I still find great profit in reading Archdeacon Paley's "Natural Theology" and Sir Charles Bell's Bridgewater treatise. The explanations they give may have become antiquated, but the facts and contrivances they describe are still before our eyes, and the manner in which these authors place their facts before the reader has a charm and clarity which are still unexcelled. By such diversions the student will relieve the monotony of the more routine studies required of him by teachers and examiners, and qualify himself, not only for the work of life, but to take a wide and permanent interest in the welfare of his profession.

FACULTY NEWS.

On Friday evening, Dec. 3rd, Prof. Oskar Klotz addressed the first open meeting of the Medical Society in the lecture room in Hart House. The address was followed by entertainment and refreshments. The January Journal will report this address.

The Medical Society is making arrangements for a series of these open meetings to be addressed by prominent men, and we are thus assured of a successful year.

The Senior Med-at-Home will be held in the Crystal Ball Room, King Edward Hotel, February 1. Romanelli's Orchestra of 16 pieces will be present.

The first of a series of Dollar Dances will be held at U.T.S. Saturday, December 4. It will be followed by one on Saturday, December 11.

Daffydil Committee got off to a good start on Wednesday, Nov. 17, when the chairman, Tom. Belt, called the committee together. With Mr. Belt at the head we are assured of much fun and frolic.

The Medical Sick Benefit Fund is going ahead with rapid strides. There are two years to be heard from yet, but so far each year has shown advancement; with Mr. Hetherington showing a fine record of 103 for second year.

EXAMINATION ERRORS. 1ST YEAR BIOLOGY.

Orientation—The ways of olden times.

Erythrocytes—The young are born alive, but naked and blind, and are found in holes in the ground. While Leucocytes the young are born with hair on and able to see, but their home is not beneath the ground.

After secretion the cells become haggard.

Dorsal is the top part of the rabbit in its natural state.

Symphisis is the posterior extremity of the abdominal cavity. It is connected with the descending colon. In the symphisis is the pubis and attached are numerous excretory glands. It is composed of cartilage and bone and runs directly from the posterior extremity of the vertebrae column, and the caudal vertebrae.

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